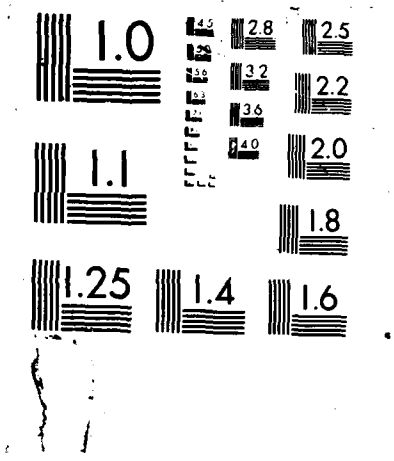
AD-A189 387 PLASMA CORTISOL RENIN AND ALDOSTERONE DURING AN INTENSE HEAT ACCLIMATION PROGRAM(U) ARMY RESEARCH INST OF ENVIRONMENTAL MEDICINE NATICK MA L E ARMSTRONG ET AL. 1/1

UNCLASSIFIED SEP 87 F/G 6/10 NL


1.0
1.1
1.25
1.4
1.6
1.8
2.0
2.2
2.5
2.8
3.2
3.6
4.0

(1)

Plasma cortisol, renin and aldosterone during an intense heat acclimation program

Lawrence E. Armstrong

Ralph P. Francesconi

William J. Kraemer

Natalie Leva

Jane P. DeLuca

Roger W. Hubbard

U.S. Army Research Institute of Environmental Medicine

Natick, Massachusetts 01760-5007

Corresponding author: Lawrence E. Armstrong, Ph.D

Heat Research Division

(617) 651-4873

Subtitle: PRA, ALD AND PC: HEAT ACCLIMATION RESPONSES

UNCLAS

SECURITY CLASSIFICATION OF THIS PAGE

REPORT DOCUMENTATION PAGE

Form Approved OMB No. 0704-0188

1a. REPORT SECURITY CLASSIFICATION	1b. RESTRICTIVE MARKINGS
2a. SECURITY CLASSIFICATION AUTHORITY: UNCLAS	3. DISTRIBUTION/AVAILABILITY OF REPORT: DISTRIBUTION A:
2b. DECLASSIFICATION/DOWNGRADING SCHEDULE	
4. PERFORMING ORGANIZATION REPORT NUMBER(S)	5. MONITORING ORGANIZATION REPORT NUMBER(S)
6a. NAME OF PERFORMING ORGANIZATION: US Army Research Institute of Environmental Medicine	6b. OFFICE SYMBOL (*If applicable*): SGRD-UE-HR
6c. ADDRESS (*City, State, and ZIP Code*): Heat Research Division, Natick, Massachusetts 01760-5007	7a. NAME OF MONITORING ORGANIZATION
	7b. ADDRESS (*City, State, and ZIP Code*)
8a. NAME OF FUNDING/SPONSORING ORGANIZATION	8b. OFFICE SYMBOL (*If applicable*)
	9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER
8c. ADDRESS (*City, State, and ZIP Code*)	10. SOURCE OF FUNDING NUMBERS

PROGRAM ELEMENT NO.	PROJECT NO.	TASK NO.	WORK UNIT ACCESSION NO.
	A879		129

11. TITLE (*Include Security Classification*)
Plasma cortisol, renin and aldosterone during an intense heat acclimation program.

12. PERSONAL AUTHOR(S)
L.E. Armstrong, R.P. Francesconi, W.J. Kraemer, N. Leva, J.P. DeLuca, R.W. Hubbard

13a. TYPE OF REPORT	13b. TIME COVERED	14. DATE OF REPORT (*Year, Month, Day*)	15. PAGE COUNT
Manuscript	FROM _____ TO _____	September 1987	19

16. SUPPLEMENTARY NOTATION

17. COSATI CODES			18. SUBJECT TERMS (*Continue on reverse if necessary and identify by block number*)
FIELD	GROUP	SUB-GROUP	plasma volume, exercise, rectal temperature, heart rate, skin temperature.

19. ABSTRACT (*Continue on reverse if necessary and identify by block number*)

The effects of an intense, intermittent heat acclimation (HA) regimen (treadmill, 41.2°C, 8 days, 100 min·d^{-1}) on stress and fluid balance hormones responses were examined in 13 unacclimated male volunteers. Venous blood samples were collected before (PRE) and after (POST) exercise (days 1, 4, 8) and analyzed for plasma renin activity (PRA), aldosterone (ALD), cortisol (PC), plasma volume shifts (ΔPV%), sodium (Na+) and potassium (K+). Subjects exhibited physiological adaptations (day 1 vs day 8) typical of HA ($p<.05$): decreased heart rate, rectal temperature, skin temperature, and improved defense of PV. While plasma Na+ demonstrated no change during daily exercise, K+ ($p<.01$), PC, PRA and ALD increased ($p<.05$) more than ΔPV% (day 1: -7.1%, day 8: -5.1%) accounted for. PRA and ALD did not change as a result of HA, but PRE vs POST PC responses were attenuated. The dissociation of PRA and ALD levels on day 4 of HA (POST) may be explained by differences in splanchnic clearance mechanisms. It was concluded that during an intense HA regimen, electrolyte and hormonal responses to exercise in the heat are modulated by the acquisition of acclimation.

20. DISTRIBUTION/AVAILABILITY OF ABSTRACT	21. ABSTRACT SECURITY CLASSIFICATION
☐ UNCLASSIFIED/UNLIMITED ☐ SAME AS RPT ☐ DTIC USERS	UNCLAS
22a. NAME OF RESPONSIBLE INDIVIDUAL	22b TELEPHONE (*Include Area Code*) / 22c. OFFICE SYMBOL

DD Form 1473, JUN 86 *Previous editions are obsolete.*

SECURITY CLASSIFICATION OF THIS PAGE

UNCLAS

Abstract

The effects of an intense, intermittent heat acclimation (HA) regimen (treadmill, 41.2°C, 8 days, 100 $min \cdot d^{-1}$) on stress and fluid balance hormones responses were examined in 13 unacclimated male volunteers. Venous blood samples were collected before (PRE) and after (POST) exercise (days 1, 4, 8) and analyzed for plasma renin activity (PRA), aldosterone (ALD), cortisol (PC), plasma volume shifts (ΔPV%), sodium (Na+) and potassium (K+). Subjects exhibited physiological adaptations (day 1 vs day 8) typical of HA ($p<.05$): decreased heart rate, rectal temperature, skin temperature, and improved defense of PV. While plasma Na+ demonstrated no change during daily exercise, K+ ($p<.01$), PC, PRA and ALD increased ($p<.05$) more than ΔPV% (day 1: -7.1%, day 8: - 5.1%) accounted for. PRA and ALD did not change as a result of HA, but PRE vs POST PC responses were attenuated. The dissociation of PRA and ALD levels on day 4 of HA (POST) may be explained by differences in splanchnic clearance mechanisms. It was concluded that during an intense HA regimen, electrolyte and hormonal responses to exercise in the heat are modulated by the acquisition of acclimation.

Index Terms

plasma volume, exercise, rectal temperature, heart rate, skin temperature

A-1

Introduction

The metabolic responses to continuous, intense exercise are different from those of intermittent, intense exercise (3). Continuous high intensity exercise is characterized by a significant carbohydrate oxidation, a rapid depletion of muscle glycogen, and lactic acid accumulation. Equivalent high intensity exercise, performed in bouts separated by short rest periods, involves an increased lipid contribution to oxidative metabolism (9) and is similar to continuous, moderate exercise rather than brief intense exercise (26). The hormonal responses to continuous and intermittent exercise also vary, especially concerning responses of fluid balance and stress hormones (10,20,23). The effects of acute heat exposure on fluid regulatory hormones have been described (18), but the effects of repeated days of exercise in the heat (heat acclimation) are uncertain due to inconsistent findings. For example, Finberg et al. twice reported that increments in plasma renin activity (PRA) stimulated by exercise-heat stress were attenuated by a 7-day heat acclimation program (12,13). However, Davies (7), Cochrane (5) and Convertino (6) observed that increases in PRA during exercise in the heat were unaffected by heat acclimation; increases in aldosterone (ALD) also were unaffected by heat acclimation (5,6,7,). Francesconi (15) explained these discrepancies by noting that differences in hydration status, exercise mode and intensity, and physical fitness of subjects may affect the direction and magnitude of body fluid shifts and hormonal responses during exercise in a hot environment. In contrast, the effects of heat acclimation on the magnitude of plasma cortisol (PC) responses have been the focus of only two investigations, to our knowledge. These investigations (16, 17) demonstrated no effects of exercise in the heat or heat acclimation on PC levels when subjects exercised intermittently at approximately 25% $\dot{V}O_2$ max and were euhydrated.

Although previous studies have examined hormonal responses to prolonged continuous exercise (23), brief intense exercise (10), and intermittent intense exercise (20) in cool environments, as well as mild intermittent exercise in the heat (15,16,17), no previous work has focused on hormonal responses to intense intermittent exercise in a hot environment (18). Such research may be relevant to industrial workers and athletic (e.g. soccer, interval running) participants. Further, few previous studies have examined intermittent high intensity exercise as a means of inducing the adaptations of heat acclimation (i.e. increased plasma volume, decreased heart rate and rectal temperature). Therefore, the following data are presented to assess the PRA, ALD and PC responses of 13 males during an 8-day heat acclimation protocol. These data describe HA adaptations to high intensity, intermittent exercise, the control of body fluid composition and movements, as well as the general stress responses experienced by subjects during daily exercise in a hot environment.

Methods

The subjects of this investigation were 13 unacclimated, healthy males with the following characteristics (mean $\pm$ SE_m): age - 28.2 $\pm$ 2.1 yr, height - 176 $\pm$ 2 cm, weight - 77.41 $\pm$ 3.20 kg, $\dot{V}O_2max$ - 46.9 $\pm$ 2.1 $ml \cdot kg^{-1} \cdot min^{-1}$. Subjects were informed of all procedures and risks prior to participating. Written consent was secured under the auspices of the local Human Use Review Board. One day prior to the beginning of heat acclimation, subjects performed a maximal oxygen consumption ($\dot{V}O_2max$) test, as described by McArdle et al (22).

The heat acclimation (HA) regimen consisted of eight days of treadmill exercise in an environmental chamber maintained at 41.2 $\pm$ 0.5°C db, 39.0 $\pm$ 1.7% rh, and 0.1 $\pm$ 0.1 $m \cdot s^{-1}$ wind velocity. Subjects ran (treadmill, mean

speed 2.7 ± 0.05 $m.sec^{-1}$, 0 angle, 68 ± 1%VO_2max) during nine exercise periods (5, 8 or 10 min duration) and stood for 2, 5 or 10 min of rest between exercise bouts. Exercise on day 1 was begun at a reduced rate to reduce the possibility of syncope. Trials on days 1 and 8 of HA were identical, but different from all other days, and consisted of walking during the first four exercise bouts and running during exercise periods 5 - 9. Trials on days 2 - 7 involved running during each period. These procedures were previously described in detail (2) and are summarized in Table 1. Because of the differences of days 1 and 8 from all other days, the day 4 results must be interpreted with the understanding that energy expenditure and physiological strain were greater on day 4 than on days 1 and 8. For example, the mean distance run on days 2 - 7 was 8.7 km, while that of days 1 and 8 was 6.3 km (Table 1).

TABLE 1

Subjects ran in pairs, one subject on each of two treadmills. Oxygen consumption and minute ventilation were measured with a semi-automated system consisting of a gas meter, oxygen analyzer, carbon dioxide analyzer, digital voltmeter, scanner and computer. Treadmill belt speeds were carefully monitored during each of 936 work periods (8 days, 9 work periods, 13 subjects) using a hand-held digital tachometer. Heart rate (HR) was monitored continuously using an ECG telemetry unit. Each subject was equipped with a rectal probe (inserted 8 cm beyond the anal sphincter) and three skin probes placed on the chest, forearm and calf. Rectal temperature (Tre) and mean weighted skin temperature (Tsk) calculations were recorded every four minutes. Exercise was terminated if heart rate exceeded 180 beats· min^{-1} during exercise, if heart rate did not fall below 160 beats· min^{-1} during rest, if rectal temperature exceeded 39.5°C, or if symptoms of heat illness (i.e. dizziness, chills, throbbing headache) warranted termination. Water was drunk

ad libitum throughout all trials, and subjects were instructed to consume large quantitites of water when they were not in the climatic chamber. Body weight (± 10g) and entering urine specific gravity were used to assess fluid status. If any subject entered a trial with a urine specific gravity greater than 1.030, he did not begin until he drank sufficient water to produce a urine sample of specific gravity below 1.030.

A 20 min standing equilibration period in the heat preceded each pre-exercise antecubital venous blood sample. A second sample was drawn immediately post exercise. Blood samples (days 1, 4, 8 only) were analyzed for hematocrit and hemoglobin (Boehringer/Mannheim, Indianapolis IN), and changes in plasma volume (ΔPV%) were calculated using hematocrit and hemoglobin values (8). Plasma Na+ and K+ were measured via flame photometry. The remainder of the blood was transferred to iced heparinized tubes and centrifuged (4°C, 10,000g), after which the plasma was removed, frozen (-20°C) and stored for subsequent assay. After thawing, PRA, ALD and PC were quantitated by use of commerically available radioimmunoassay test kits (PRA and PC - New England Nuclear, North Billrica, MA; ALD - International CIS, Saluggia, Italy), utilizing procedures outlined in their technical bulletins. Normal ranges for these hormones have been reported as follows: PRA - 0.5-4.0ng·ml^{-1}·h^{-1} for adult normotensive subjects, ALD - 7-29.5ng·100ml^{-1} for upright subjects, and PC - 4.2-25ug·100ml^{-1}.

Dietary records were maintained by all subjects, in an effort to estimate the impact of dietary intake on body weight, plasma Na+, plasma K+ and ALD. Subjects recorded the quantity, type, brand name and method of preparation for all food and beverages consumed on days 1 - 3 and days 6 - 8 of HA. Dietary records were reviewed for completeness and accuracy by personal daily interview. Estimates of caloric intake, Na+ content, and K+ content were made with the aid of a the U.S. Department of Agriculture Handbook (28).

ANOVA were utilized to statistically analyze hormone levels, plasma Na+ and K+, daily entering body weight, and urine specific gravity. The Student-Neuman-Keuls post hoc analysis was applied when significance was indicated. Paired t-tests were used to compare final HR, ΔHR, final Tre, ΔTre, final Tsk, and ΔTsk on days 1 and 8. All data were expressed mean ± the standard error of the mean; the null hypothesis was rejected at $p < .05$.

Results

No significant between-day differences ($p>.05$) were found when pre-exposure body weight and urine specific gravity were compared on days 1 through 8; therefore, subjects were considered to be in a euhydrated state at the beginning of each trial. Dietary records indicated no significant between-day differences ($p>.05$) in caloric consumption, Na+ intake, or K+ intake, thus allowing us to conclude that diet had little or no impact on hormone levels depicted in Figure 1.

FIGURE 1

To assess the physiological adaptations which resulted from 8 days of intense, intermittent running in the heat, variables were compared on day 1 and day 8, and the following indices were significantly ($p<.05$) reduced over the acclimation period: final HR (170 ± 3 vs 144 ± 5 beats·min $^{-1}$), ΔHR (84 ± 3 vs 68 ± 6 beats·min $^{-1}$), final Tre (39.17 ± 0.10 vs 38.52 ± 0.16 ^{o}C), ΔTre (2.04 ± 0.09 vs 1.46 ± 0.18^{o}C), final Tsk (37.58 ± 0.23 vs 36.53 ± 0.29 ^{o}C), ΔTsk (1.68 ± 0.21 vs 1.29 ± 0.40 ^{o}C), and within-day ΔPV% (-7.1 ± 0.9 vs -5.1 ± 1.1 %). The latter measurement (ΔPV%) indicated a better defense of plasma volume during treadmill running as a result of HA. Thus, all subjects were fully acclimated as a result of this heat/exercise regimen.

Plasma Na+ (PRE vs POST) values (mEq·L^{-1}) were as follows: day 1 - 141 ± 1 vs 140 ± 1, day 4 - 141 ± 1 vs 140 ± 1, day 8 - 140 ± 1 vs 141 ± 1; thus, no significant between-day or within-day differences were observed. Plasma K+

(PRE vs POST) values ($mEq \cdot L^{-1}$) were: day 1 - 4.3 $\pm$ 0.1 vs 4.7 $\pm$ 0.1, day 4 - 4.5 $\pm$ 0.1 vs 4.7 $\pm$ 0.1, day 8 - 4.4 $\pm$ 0.1 vs 4.8 $\pm$ 0.1; no significant between-day differences were found, but all within-day plasma K+ values were significantly increased ($p<.01$) by exercise in the heat. Calculations using pre-exercise hematocrit (day 1 - 45 $\pm$ 1, day 4 - 44 $\pm$ 1, day 8 - 44 $\pm$ 1) and hemoglobin ($mg \cdot dl^{-1}$) values (8) (day 1 - 16.30 $\pm$ 0.28, day 4 - 15.64 $\pm$ 0.33, day 8 - 15.74 $\pm$ 0.35) indicated a mean plasma volume expansion of +5 % during HA, as anticipated.

The effects of intermittent intense exercise in the heat on plasma hormonal responses on days 1, 4 and 8 of heat acclimation are depicted in Fig 1. It is noteworthy that on day 1 of heat acclimation the combined stress of the heat exposure and the nine exercise periods elicited a significant ($p<0.05$) elevation in mean circulating PC level. However, on day 8 of heat acclimation, the identical heat/exercise regimen did not elicit a significant pre- to post-exercise increment. It also should be noted that mean circulating PC levels (POST) exhibited a decreasing trend from day 1 through day 8, in spite of the increased strain experienced on day 4, and in contrast to the trends observed in mean post-exercise ALD levels on days 1, 4 and 8.

This intermittent, high intensity exercise protocol induced significant ($p<.001$) within-day increments in both ALD and PRA levels (Fig. 1) on each of the heat acclimation days, which were far greater than could be explained by hemoconcentration ($\Delta PV\% = -7.1\%$ and -5.1%). The between-day PRA responses (day 1 vs 8) to exercise in the heat were not significantly different. On days 4 and 8, mean baseline levels of ALD were approximately twice the day 1 levels, though not significantly different ($p>.05$). The group mean ALD level (POST) measured on day 4 was significantly greater than POST values on days 1 and 8, probably reflecting the greater total work completed on day 4, in contrast to PRA levels which were not different on day 4.

Discussion

Generally, the physiological strain of light exercise (1.3 $m \cdot s^{-1}$, 0% grade) in a hot environment (35 - 40°C) is easily tolerated by acclimated, euhydrated test subjects without significant increments in PC levels (16,17). However, we have also reported (16, 17) that both hypohydration and non-acclimation can significantly alter PC levels both prior to and during exercise in the heat. The results of the present investigation are consistent with the hypothesis that the acquisition of heat acclimation enables test subjects to tolerate exercise-heat stress at a reduced physiological cost; this reduced strain may be, in turn, reflected in the decrements which occurred in PC levels subsequent to exercise in the heat on day 8 (vs day 1). In the current investigation, the reduction in PC subsequent to exercise on day 8 is particularly noteworthy in light of the intensity of the running exercise during exercise periods 5 - 9. This reduction in the PC response to exercise/heat stress may be related to increased body water (24), electrolyte conservation (1), increased physical fitness (19), increased metabolic efficiency (14), or reduced thermoregulatory strain. Alternatively, the elevated peripheral clearance of cortisol, which may be associated with exercise in the heat (11), may mask an ACTH-stimulated increase in cortisol secretion (7).

Following exercise on each test day, levels of PRA were significantly ($p<0.001$) increased by the heat/exercise protocol. It is likely that intense exercise diverted blood away from the kidneys and increased renin secretion from juxtaglomerular cells, in response to the increased stretch of receptors in the renal vasculature. Whereas in our earlier studies (15,17) we generally observed moderate increments in PRA (range: 1 - 9 $ng \cdot ml^{-1} \cdot h^{-1}$) during light exercise in the heat, the exercise in the current experiments was more intense and the mean levels of PRA were proportionately higher (range:

9.7-12.7ng·ml^{-1}·h^{-1}). Results of the current investigation did not indicate that PRA levels were affected by either exercise intensity or the state of HA, although we observed such an effects following acclimation in a previous study, particularly in hypohydrated test subjects (15). The present results disagree with those of Finberg et al (12,13), who observed attenuation of PRA increases following HA, but agree with the results of other investigators (5,6,7) who observed no reduction of PRA levels after HA programs.

The absolute levels of ALD observed subsequent to exercise in the heat are considerably higher than we reported in our earlier studies (15,17). This is probably a direct manifestation of the increased intensity of the exercise protocol (Table 1) which was implemented in the current investigation and apparently was not related to altered dietary Na+ intake. There are two additional points that are worthy of comment. In our earlier study (15), euhydrated test subjects manifested higher pre-exercise levels of ALD following acclimation; in the current study, the mean pre-exercise level on Day 8 (28.2 ng·100 ml^{-1}) was markedly higher than the comparable level on Day 1 (15.7 ng·100 ml^{-1}). Also, HA did not attenuate the ALD response to exercise in the heat in our earlier report (15), and in the present experiment the post-exercise mean ALD level on Day 8 (87.2 ng·100 ml $^{-1}$) was not different (NS) from that observed on Day 1 (80.0 ng·100 ml^{-1}). Thus, these data indicate that the intensity of the ALD response may be correlated with the intensity of the exercise, that consecutive days of exercise in the heat may result in higher baseline levels of ALD, and that heat acclimation does not decrease the ALD (POST) response to exercise in the heat. This latter observation agrees with results published by four other research groups (5,6,7,13).

Dissociation of the responses of PRA and ALD levels have been reported previously during exercise (21), during acute heat exposure (4), and subsequent to HA (12,15). The data presented in Figure 1 also demonstrate a dissociation of PRA and ALD responses, in measurements following exercise in the heat on day 4. In comparison to day 1, the POST day 4 PRA decreased but ALD increased considerably. It is possible that this dissociation resulted from differences in the turnover rates of PRA and ALD. In dogs (27), it has been demonstrated that hepatic clearance of ALD is nearly 100% and that any reduction in blood flow to the liver produces a decrease in hepatic extraction; consequently, the metabolism of ALD is controlled by a flow-limited system. However, renin clearance is actually increased as hepatic blood flow is decreased in dogs; the mechanism for this compensatory elevation in renin removal is unknown (27). If analogous mechanisms exist in humans, the dissociation of PRA and ALD levels (day 4) in Figure 1 can be explained, especially since splanchnic blood flow is greatly reduced by intense exercise in a hot environment (25).

In summary, because within-day PC levels indicated significant ($p<.05$) increases on days 1 and 4 (13.6 vs 20.0 and 10.9 vs 16.8, respectively) but not on day 8, HA apparently reduced the overall stress on these subjects, which was also manifested in the significant ($p<.05$) decreases in HR, Tre and Tsk during HA. Alternatively, PRA and ALD significantly ($p<.05$) increased during exercise on all days, but PRA and ALD exhibited no attenuation (day 1 vs day 8) of POST exercise increases. Pre-exercise ALD levels, unaffected by dietary Na^+ consumption, on day 4 and 8 approximately doubled (in comparison to day 1) as a result of HA. The dissociation of PRA and ALD levels on day 4 (Fig 1) may be the result of differences in clearance of PRA and ALD by the liver, during a period of reduced splanchnic blood flow (e.g. intense exercise in the heat).

Acknowledgements

The authors gratefully acknowledge the technical assistance of Dr. Patricia C. Szlyk, Ingrid V. Sils, Susan E.P. Henry and Diane Danielski.

The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as an official department of the Army position, policy, or decision, unless so designated by other official documentation. Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

References

1. Allan, J.R., and C.G. Wilson. Influence of acclimatization on sweat sodium concentration. J. Appl. Physiol. 30: 708-712, 1971.

2. Armstrong, L.E., R.W. Hubbard, J.P. DeLuca, and E.L. Christensen. Self-Paced Heat Acclimation Procedures. Natick, MA: USARIEM Technical Report No. T8-86, 1986, p. 1-28.

3. Astrand, I., P-O. Astrand, E.H. Christensen, and R. Hedman. Intermittent muscular work. Acta Physiol. Scand. 48:448-453, 1960.

4. Brandenberger, G., M. Follenius, and S. Oyono. Effect of propranolol on aldosterone response to acute heat exposure in sodium-restricted men. J. Endocrinol. Invest. 4:395-400, 1980.

5. Cochrane, L.A., J. A. Davies, R.J. Edwards, and M.H. Harrison Some adreno-cortical responses to heat acclimatization. J. Physiol. 30:32-33P, 1979.

6. Convertino, V.A., J.E. Greenleaf, and E. M. Bernauer. Role of thermal and exercise factors in the mechanism of hypervolemia. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 48:657-664, 1980.

7. Davies, J.A., M.H. Harrison, L.A. Cochrane, R.J. Edwards, and T. M. Gibson. Effects of saline loading during heat acclimation on adrenocortical hormone levels. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 50:605-612, 1981.

8. Dill, D.B., and D.L. Costill. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. J. Appl. Physiol. 37:247-248, 1974,

9. Essen, B. Studies on the regulation of metabolism in human skeletal muscle using intermittent exercise as an experimental model. Acta Physiol. Scand. Suppl 454:1-33, 1978.

10. Farrell, P.A., T.L. Garthwaite, and A.B. Gustafson. Plasma adrenocorticotropin and cortisol responses to submaximal and exhaustive exercise. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 55:1441-1444, 1983.

11. Few, J.D. Effect of exercise on the secretion and metabolism of cortisol in man. J. Endocrinol. 62:341-353, 1974.

12. Finberg, J.P.M., M. Katz, H. Gazit, and G.M. Berlyne. Plasma renin activity after acute heat exposure in nonacclimatized and naturally acclimatized man. J. Appl. Physiol. 36:519-523, 1974.

13. Finberg, J.P.M., and G.M. Berlyne. Modification of renin and aldosterone response to heat by acclimation in man. J. Appl. Physiol.: Respirat. Envion. Exercise Physiol. 42:554-558, 1977.

14. Fink, W.J., D.L. Costill, and P.J. Van Handel. Leg muscle metabolism in the heat and cold. Eur. J. Appl. Physiol. 34:183-190, 1975.

15. Francesconi, R.P., M.N. Sawka, and K.B. Pandolf. Hypohydration and heat acclimation: plasma renin and aldosterone during excercise. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 55:1790-1794, 1983.

16. Francesconi, R.P., M.N. Sawka, and K.B. Pandolf. Hypohydration and acclimation: effects on hormone responses to exercise/heat stress. Aviat. Space Environ. Med. 55:365-369, 1984.

17. Francesconi, R.P., M.N. Sawka, K.B. Pandolf, R.W. Hubbard, A.J. Young, and S. Muza. Plasma hormonal responses at graded hypohydration levels during exercise-heat stress. J. Appl. Physiol. 59: 1855-1860, 1985.

18. Galbo, H. Hormonal and Metabolic Adaptation to Exercise. Stuttgart: Thieme-Stratton Inc., 1983, p. 1-116.

19. Gisolfi, C.V., and J.S. Cohen. Relationships among training, heat acclimation, and heat tolerance in men and women: the controversy revisited. Med. Sci. Sports 11:56-59, 1979.

20. Kosunen, K.J., and A.J. Pakarinen. Plasma renin, angiotensin II, and plasma and urinary aldosterone in running exercise. J. Appl. Physiol. 41:26-29, 1976.

21. Kosunen, K., A.J. Pakarinen, A. Kuoppasalmi, H. Nareri, S. Rehunen, C.G. Slanderskjold-Nordenstan, M. Harkonen, and H. Adlercreutz. Cardiovascular function and the renin-aldosterone system in long distance runners during various training periods. Scand. J. Clin. Lab. Invest. 40:429-435, 1980.

22. McArdle, W.D., F.I. Katch, and G.S. Pechar. Comparison of continuous and discontinuous treadmill and bicycle tests for max VO_2. Med Sci. Sports 5:156-160, 1973.

23. Newmark, S., T. Himathongkam, R. Martin, K. Cooper, and L. Rose. Adrenocortical reponse to marathon running. J. Clin. Endocrinology Metabolism 42:393-394, 1976.

24. Oddershede, I.R., and R.S. Elizondo. Body fluid and hematologic adjustments during resting heat acclimation in rhesus monkey. J. Appl. Physiol.: Respirat Environ. Exercise Physiol. 49:431-437, 1980.

25. Rowell, L.B. Human cardiovascular adjustments to exercise and thermal stress. Physiol. Rev. 54:75-159, 1974.

26. Saltin, B., and B. Essen. Muscle glycogen, lactate, ATP and CP in intermittent exercise. In: Muscle Metabolism During Exercise. New York: Plenum Press, 1971, p. 419-424.

27. Schneider, E.G., J.O. Davis, J.S. Baumber, and J.A. Johnson. The hepatic metabolism of renin and aldosterone. Circ. Res. 26-27 (suppl. 1): I-175 - I-183, 1970.

28. U.S. Department of Agriculture. Composition of Foods. Washington D.C.: U.S. Government Printing Office, 1975, p. 1-190.

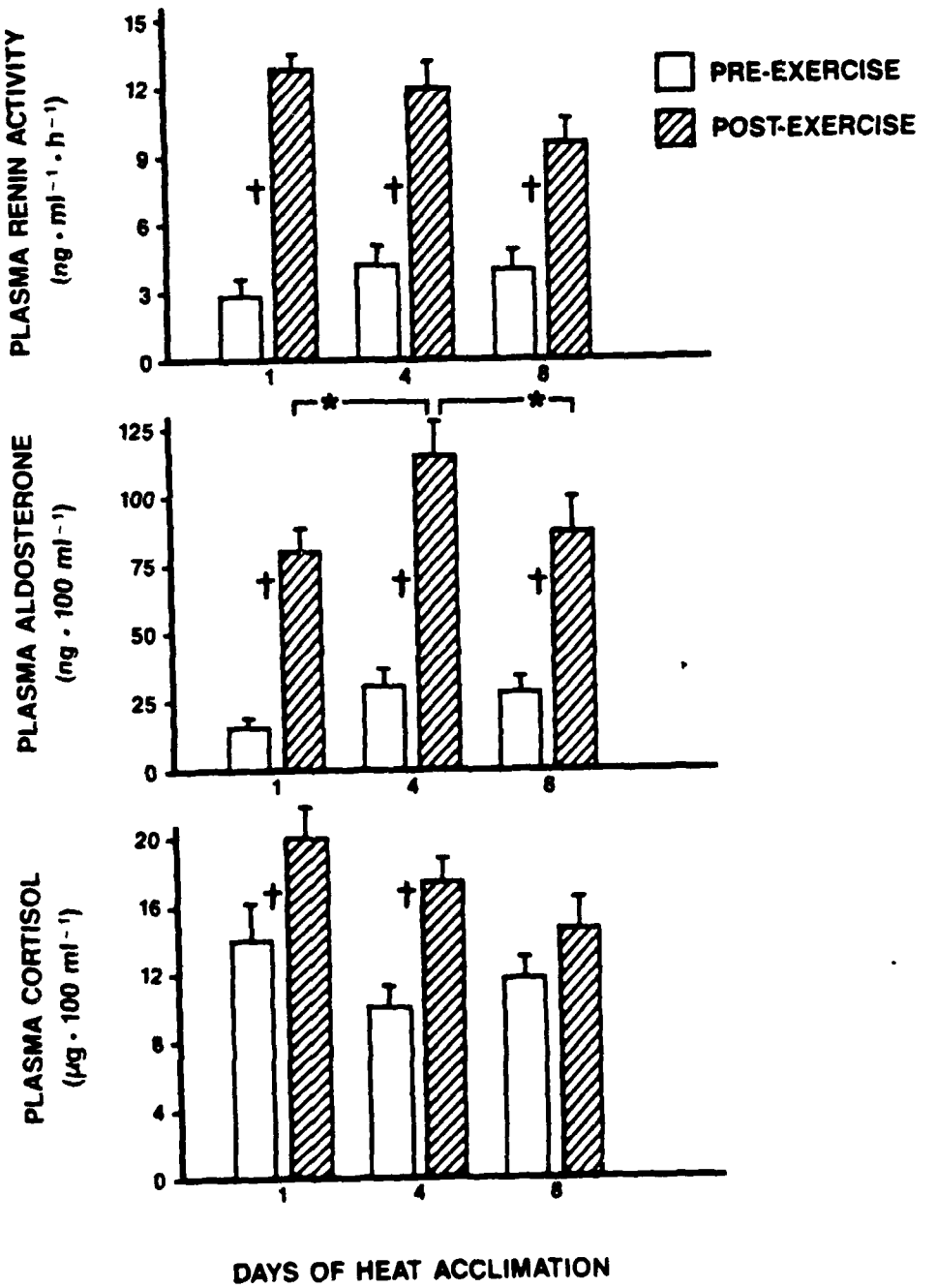
Table 1 - Duration of Exercise-Rest Periods During Heat Acclimation Trials, Days 1 - 8.

Period	Duration (min)
Exercise 1	5 *
Rest 1	2
Exercise 2	8 *
Rest 2	2
Exercise 3	8 *
Rest 3	5
Exercise 4	5 *
Rest 4	10
Exercise 5	10
Rest 5	5
Exercise 6	5
Rest 6	5
Exercise 7	5
Rest 7	5
Exercise 8	5
Rest 8	5
Exercise 9	5

* - Exercise periods involved running at 68 ± 1 % $\dot{V}O_2max$, except periods 1 - 4 on days 1 and 8, during which subjects walked at 0.95 $m \cdot sec^{-1}$ (periods 1 - 2) and at 1.58 $m \cdot sec^{-1}$ (periods 3 - 4), and then ran during periods 5 - 9.

Figure Legend

Figure 1 - Effects of 8 days of intense intermittent treadmill exercise on PRA, ALD and PC levels in a hot-dry environment (41.2°C, 39 % rh). Mean (± SE) values are depicted for n = 13 on all days. Blood samples were taken after standing for 20 min in the heat (pre-exercise) and immediately following exercise period 9 (post-exercise). Days 1 and 8 were identical trials, in which subjects walked for 4 periods and ran for 5 periods. Day 4 involved running for 9 periods (see Table 1). All within-day measurements of PRA and ALD were significantly different (†) at the $p < .001$ level. Within-day measurements of PC (pre- vs post-exercise) were significantly different (†) at the $p < .05$ level on day 1 and day 4, but not on day 8 (NS). No between-day differences were observed in either pre-exercise or post-exercise values of PC or PRA. Statistically significant between-day differences for ALD were observed only in post-exercise samples and are represented as * ($p < .05$).


PRE-EXERCISE
POST-EXERCISE
PLASMA RENIN ACTIVITY
(ng • ml⁻¹ • h⁻¹)
15
12
9
6
3
0
†
†
†
1
4
8
*
*
PLASMA ALDOSTERONE
(ng • 100 ml⁻¹)
125
100
75
50
25
0
†
†
†
1
4
8
PLASMA CORTISOL
(µg • 100 ml⁻¹)
20
16
12
8
4
0
†
†
1
4
8
DAYS OF HEAT ACCLIMATION

END

DATE

FILMED

MARCH

1988

DTIC